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Technical Note

Use of the Cross-Over Buddy Wire Technique for Coronary Stent Navigation to the Basilar Artery in Acute Ischemic Stroke Due to Basilar Artery Atherosclerotic Occlusion

Tadao Shinohara, Yasutaka Yokoi, Junichi Kubota, and Shigeo Mukaihara

Objective: Several techniques have been reported for navigating devices to the basilar artery (BA). We report a case of acute BA reconstruction using the buddy wire technique to guide a coronary stent to the BA.

Case Presentation: The patient was a 74-year-old man without a history of stroke. He suddenly developed quadriplegia and coma due to basilar artery occlusion (BAO). Although mechanical thrombectomy with a stent retriever and subsequent balloon angioplasty were performed repeatedly for residual severe stenosis, recanalization was not maintained. Recanalization with a coronary stent was attempted, but guidance was difficult because of the tortuous vertebral artery (VA). The stent was successfully guided to the BA by navigating two microguidewires as far as possible to the contralateral VA across the union.

Conclusion: The cross-over buddy wire technique is a useful option for guiding a coronary stent to the BA.

Keywords ▶ buddy wire technique, acute ischemic stroke, coronary stent, tortuous, basilar artery occlusion

Introduction

In revascularization of acute arteriosclerosis obliterans in the posterior circulation, cases in which recanalization cannot be maintained by balloon dilatation alone and stent placement is necessary are common.[1–3] In Japan, no device is covered by national health insurance. A coronary arterial stent may be used, but its use is off-label and navigation is difficult because it is a rapid-exchange type. We report a patient with acute basilar artery occlusion (BAO) in whom a coronary arterial stent was unable to be navigated across the vertebral artery (VA) during the atlantoaxial transition, but a coronary arterial stent was successfully navigated into the cranium by employing the cross-over buddy wire technique[4] to guide two wires to the contralateral VA beyond the VA junction.

Department of Neurosurgery, Fujisawa City Hospital, Fujisawa, Kanagawa, Japan

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Corresponding author: Tadao Shinohara. Department of Neurosurgery, Fujisawa City Hospital, 2-6-1, Fujisawa, Fujisawa, Kanagawa 251-8550, Japan
Email: tashino9@yahoo.co.jp

Case Presentation

Patient: A 74-year-old male
Chief complaints: Quadriplegia and disturbance of consciousness
Past medical history: None
Familial medical and social histories: He had previously smoked 40 cigarettes a day but quit.
History of present illness: Transient weakness of the extremities developed 12 days before onset. He visited the emergency department of our hospital for chief complaints of transient dysarthria and numbness of the right hand, and detailed outpatient examination was scheduled one day prior. In January 2018, the patient was confirmed to be healthy at 6:00. His consciousness became disturbed at home at 9:00 and he was transported to our hospital by ambulance (3 hours and 57 minutes from the final pre-disease state to transport to our hospital).
Status on admission: The consciousness level was Japan Coma Scale 200 and Glasgow Coma Scale 4 (Eye 1 Verbal 1 Motor 2), the pupil was 3.0 mm on the right and 3.0 mm on the left, and the position of the eye was fixed at the

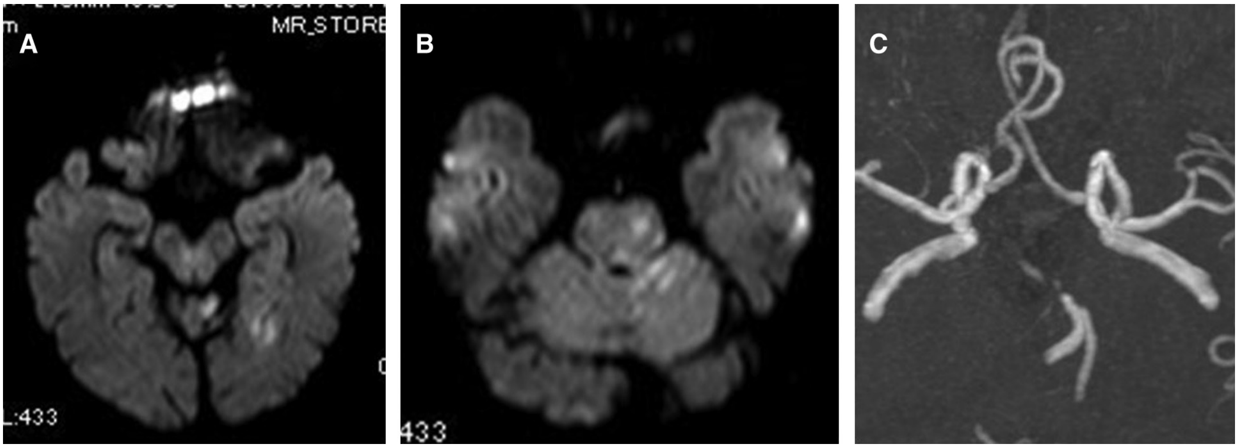

Fig. 1 Pretreatment CT and MRI imaging. (**A** and **B**) Diffusion-weighted images show acute ischemic stroke partially in the pons, left cerebellar hemisphere, and left occipital lobe. (**C**) MRA before thrombectomy shows BAO. BAO: basilar artery occlusion; CT: computed tomography; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging

median on both sides. The National Institutes of Health Stroke Scale (NIHSS) score was 40.

Test findings on admission: No major abnormal value was noted on blood chemistry. On electrocardiography, the pulse was 47 with sinus rhythm. A low-density area was present in the left pons on plain head computed tomography (CT).

On diffusion-weighted magnetic resonance imaging (MRI) of the head, pale high-intensity areas were detected in the left pons, left cerebellar hemisphere, and a part of left occipital lobe, and the posterior circulation Acute Stroke Prognosis Early CT score[5] was 7 (**Fig. 1A** and **1B**). On head magnetic resonance angiography (MRA), the basilar artery (BA) was occluded in the proximal region (5 hours and 38 minutes from the final pre-disease state to MRI) (**Fig. 1C**).

Course: The ischemic range assumed from the occluded blood vessel was deviated from the infarction range on diffusion-weighted imaging. As arrhythmia was not observed and he had a history of heavy smoking, arteriosclerotic BAO-induced brainstem infarction was considered. Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) is not applied because of the time-out. As severe nervous system disorder was observed, percutaneous thrombectomy was immediately performed to improve the symptoms.

A 6F long sheath was placed in the right femoral artery under local anesthesia (7 hours and 42 minutes from the final pre-disease state to puncture). In addition to systemic heparinization, a loading dose of 200 mg of aspirin and 300 mg of clopidogrel through a nasogastric tube was administered. On preceding diagnostic cerebral angiography, the right posterior cerebral artery (PCA) was visualized via the posterior communicating artery on internal carotid arteriography, but the BA tip was absent. The left posterior communicating artery was hypoplastic. Severe stenosis was noted in the left subclavian artery on left subclavian arteriography, and the left VA was not visualized (**Fig. 2A**). After diagnostic cerebral angiography, thrombectomy treatment was planned. When a 6F FUBUKI (Asahi Intecc, Aichi, Japan) was guided to the right VA and imaged, the BA was occluded in the distal region of the VA branching into the right anterior inferior cerebellar artery immediately after joining, and the subclavian steal phenomenon was observed (**Fig. 2B**). As acquisition was difficult due to respiratory variation, anesthesia was switched to general anesthesia. The mechanism of arteriosclerosis obliterans was considered at the beginning, considering the possibility of arteriogenic cerebral embolism from the contralateral subclavian artery stenosis lesion and recanalization using a stent retriever was performed. A Trevo pro18 microcatheter (Stryker, Kalamazoo, MI, USA) was guided to the right PCA using a Chikai 014 (Asahi Intecc) as the axis. By the first recovery technique using a Trevo XP 4.0/20 mm (Stryker), proximal occlusion of the BA tip was recanalized with residual stenosis, but BA tip occlusion remained. The second recovery technique was applied to the right PCA over the BA using the Trevo XP, and recanalization of thrombolysis in cerebral infarction (TICI) 2B was achieved, with distal occlusion of the left PCA remaining (56 minutes from puncture to recanalization and 8 hours and 38 minutes from the final pre-disease state). The third Trevo XP recovery technique was additionally applied to the residual thrombus in the lesion, followed by balloon dilatation of the lesion (**Fig. 2C**). Using the Chikai14 as an axis, the stenosis region was divided into two regions using

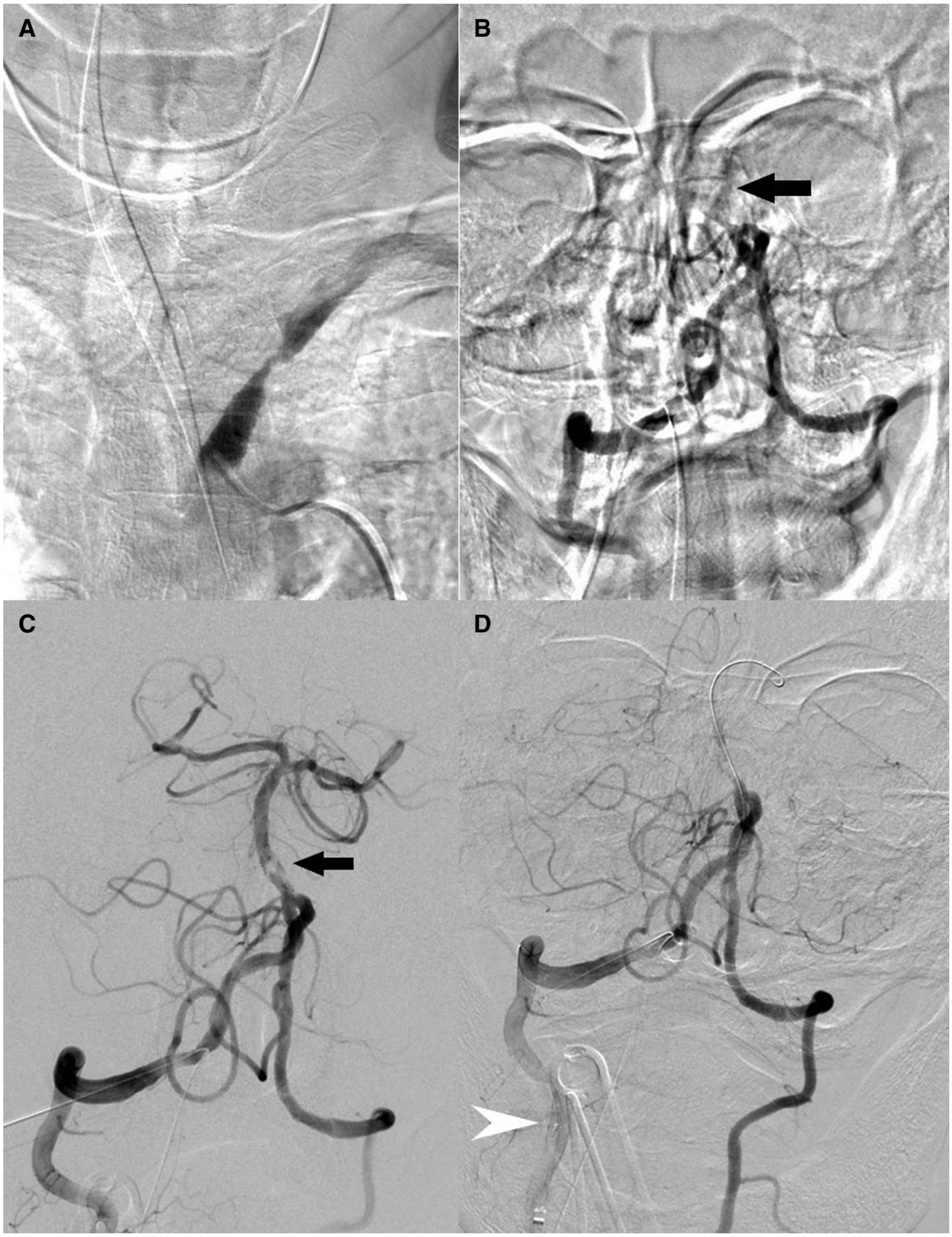

Fig. 2 Mechanical thrombectomy via the right VA. (**A**) Left subclavian angiography shows severe left subclavian artery stenosis and no antegrade flow in the left VA. (**B**) Pretreatment right vertebral angiography shows BA trunk occlusion (black arrow) and retrograde flow in the left VA. (**C**) Almost complete recanalization was obtained after three passes of the Trevo XP stent retriever. Dissection was suspected at residual stenotic lesion. (**D**) The BA was re-occluded during stent navigation. The white arrowhead indicates the tip of the coronary stent. BA: basilar artery; VA: vertebral artery

an Unryu XP 2. 5/10 mm (Kaneka Medix, Osaka, Japan) and dilated at nominal 6 atm. The region was recanalized once after dilatation, but it re-occluded and dilation was applied again at 6 atm. Recanalization was acquired, but patency was unable to be maintained. Thus, dilation using a coronary arterial stent, Integrity 3. 0/15mm (Medtronic, Minneapolis, MN, USA) was planned. As use of this stent is off-label, it was employed after receiving sufficient informed consent from the patient's family and approval by the ethics committee. In addition, 100 mg of aspirin was administered. The stent was navigated using the Chikai placed in the distal lesion as an axis, but guiding beyond

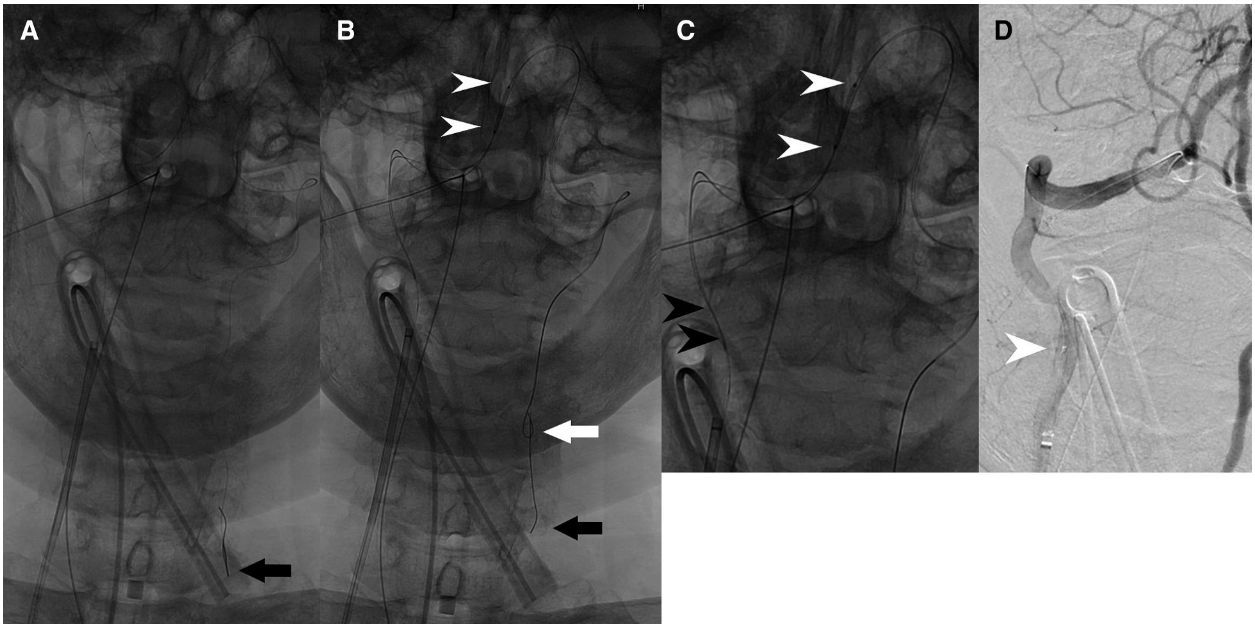

Fig. 3 (**A**) Although the Chikai black micro guidewire (black arrow) was set right-to-left across the VA, the coronary stent was unable to be navigated beyond the V2 segment. (**B**) The Chikai micro guidewire (white arrow) was set parallel to the Chikai black micro guidewire (black arrow) across the VA, and the coronary stent was navigated beyond the V2 segment (white arrowhead). (**C**) (Extended figure with **Fig. 3B**) After the cross-over buddy wire technique, the right VA was stretched (black arrowhead). (**D**) (Extended figure with **Fig. 2D**) Before the cross-over buddy wire technique, the coronary stent was unable to be navigated beyond the V2 segment (white arrowhead). VA: vertebral artery

the atlantoaxial transition area, the VA V3 segment, was not possible. Vascular extension was attempted by rotating the neck region in all directions, but the stent was unable to be navigated. The wire was changed to the harder Chikai black 014 (Asahi Intecc) and guided to the left PCA, but the stent was still unable to be navigated.

When images were acquired, stenosis had progressed and reflux had been reduced (**Fig. 2D**). Thus, balloon dilatation was additionally performed, which improved reflux. As guiding to the contralateral left VA beyond the vertebrobasilar junction was possible by pulling back the Chikai black 014, it was retrogradely guided to a site sufficiently distal to the proximal left clavicle, but the stent was unable to be navigated (**Fig. 3A**). Thus, in parallel to the Chikai black 014 guided to the left VA, a buddy wire was applied to the Chikai 014 placed beforehand to retrogradely guide the left VA to the 5th cervical vertebral level beyond the vertebrobasilar junction, which enabled extension of the VA and navigation of the stent into the cranium across the atlantoaxial transition area (**Fig. 3B–3D**). The buddy wire Chikai was removed, the Chikai black was re-guided to the cisterna ambiens, portion of the left PCA beyond the lesion, and the stent was placed and deployed in the occluded region of the BA (2 hours and 57 minutes from puncture to stent deployment) (**Fig. 4A** and **4B**). The technique was completed after confirming the maintenance of recanalization (**Fig. 4C**).

As inhibition of aggregation by the addition of cilostazol for clopidogrel insensitivity was reported to be effective after surgery,[6] in addition to 100 mg of aspirin and 75 mg of clopidogrel, 200 mg of cilostazol was added and the condition was managed by three antiplatelet drugs until the 52nd hospital day. However, adenosine diphosphate (ADP) aggregation inhibition was insufficient according to the platelet aggregation test performed on the 4th hospital day, and anticoagulant therapy using continuous heparin was additionally performed until the 12th hospital day, targeting a value double that of the previous APTT value. On the platelet aggregation test on the 17th hospital day, both collagen aggregation and ADP aggregation were inhibited. Cilostazol administration was completed on the 52nd hospital day. On head MRI after treatment, a new infarct lesion had formed on the right side of the pons in addition to those in the left thalamus and part of the right cerebellum present before treatment. Tracheotomy was performed on the 12th hospital day. On cerebral angiography performed on the 44th hospital day, the stent had acquired favorable patency (**Fig. 4D**). Paralysis of the extremities was mostly improved, but activities of daily living (ADL) had not and the patient was transferred to another hospital for rehabilitation with a modified Rankin Scale of 5 on the 57th hospital day.

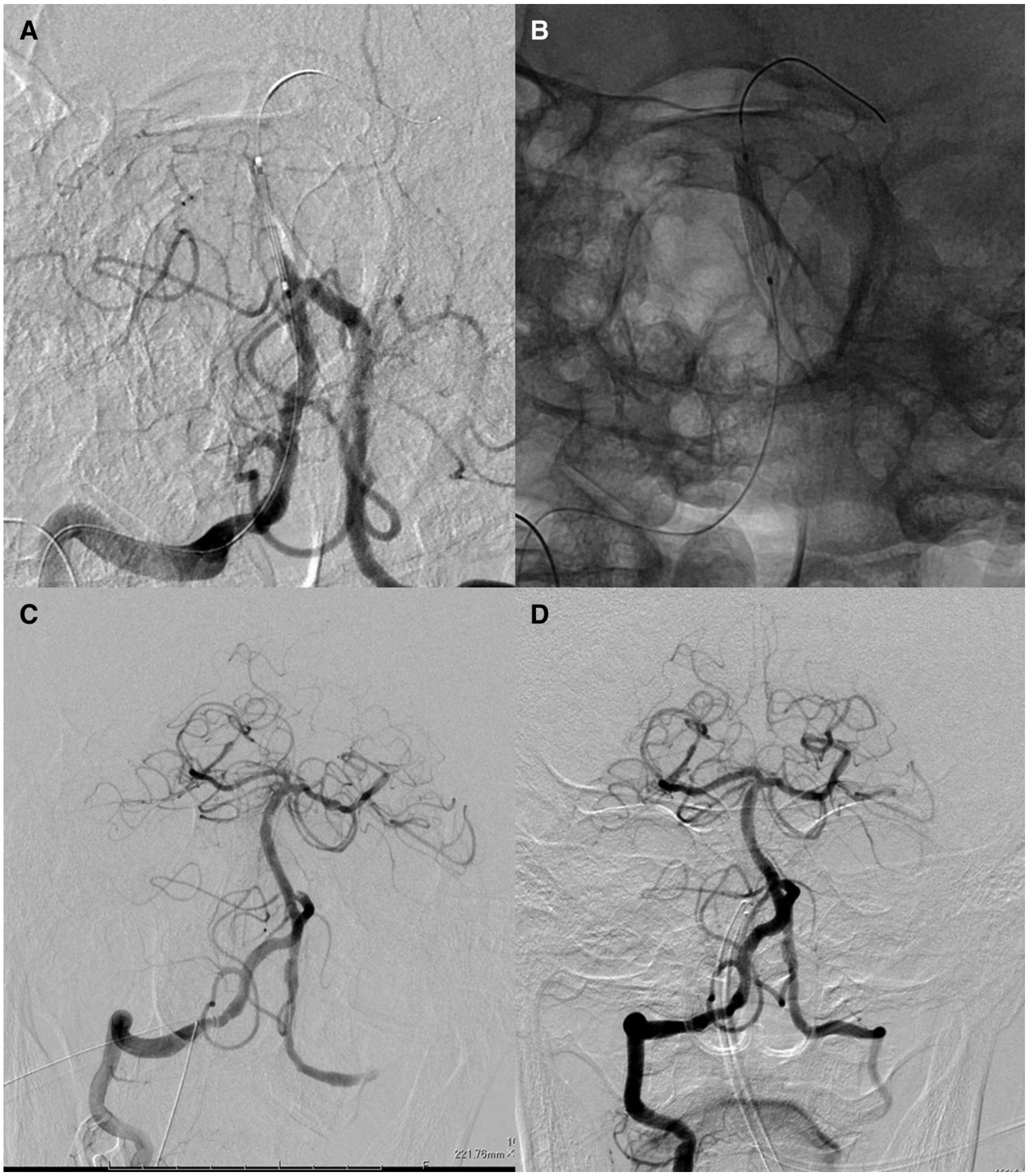

Fig. 4 (**A**) Right vertebral angiography before stent deployment. (**B**) Stent deployment to the occluded basilar trunk. (**C**) Final right vertebral angiography shows almost complete recanalization after stent angioplasty. (**D**) Right vertebral angiography 44 days after treatment shows no restenosis or re-occlusion.

Discussion

There is no standard procedure for revascularization of acute-phase cerebral infarction in the posterior circulation caused by major artery occlusion,[7] but it is not rare to encounter patients requiring acute-phase revascularization of acute arteriosclerosis obliterans of the posterior circulation by balloon dilatation and stent placement. Regarding the outcome of acute-phase revascularization for arteriosclerotic BA occlusion, Kim et al. reported that it was poor compared with that of the embolic mechanism because re-occlusion is likely due to the long time required for the technique and frequent proximal occlusion of the BA, which is likely to cause locked-in syndrome.[1] However, the outcomes were equivalent in other reports[2,3] and no specific consensus has been reached.

In the present case, patency was unable to be maintained by the stent retriever and balloon dilatation alone, and recanalization by coronary arterial stenting was attempted, but stent navigation into the cranium was difficult. Coronary arterial stenting is not covered by national health insurance and it is a rapid-exchange type, suggesting that the inappropriate structure in the cranium for treatment is the cause. As other stents, the Wingspan stent and neck bridge stent for aneurysmal coil embolization have been considered. The former has an over-the-wire-type structure and the latter has a low profile, thus guidance of these may have been relatively simple in the present case, but both are off-label and expensive. As this was an acute-phase treatment case and both devices are expensive and not always available in the hospital, the relatively low-price coronary arterial stent was selected.

The buddy wire technique was reported by Selig et al.[4] to secure a true lumen in coronary interventions. The efficacy of device navigation by vascular stretching in carotid arterial stent placement[8] and intracranial stent navigation[9] in the neuro-endovascular field was subsequently reported. In the present case, rotation of the neck region and switching to a hard wire were performed, but the stent was unable to be navigated. Moreover, the hard wire was guided to the contralateral VA to strengthen support, but guiding was not possible. Thus, via the buddy wire technique, two wires were inserted into the contralateral VA to extend the VA, enabling stent navigation into the cranium. Use of the buddy wire technique for stent navigation into the cranium for cerebral ischemia has been previously reported,[9] but its use for the posterior circulation has not been described in detail. Although the cross-over technique[10,11] for stent placement in embolization of posterior inferior cerebellar aneurysms has been reported, to our knowledge, this is the first report of the application of a buddy wire by guiding a wire to the contralateral side beyond the vertebrobasilar junction. As other guiding techniques, Rossen et al.[12] reported a hybrid retrograde antegrade approach via the posterior communicating artery for BA occlusion in which it was difficult to cross the lesion. Shin et al.[13] reported a method of retaining and pulling the wire by snaring from the contralateral VA for neck bridge stent navigation in coil embolization. This method was not applicable in the present case because the contralateral VA was not antegradely visualized, but it may be effective for acute-phase revascularization. Furthermore, switching to the transbrachial approach may have strengthened the support system. A method using an intermediate catheter is also available, but it was inapplicable because the guiding system was 6 F in this case. Supportability of the 6-F system was considered to be insufficient, but a large-diameter guidance system may not be applicable to the VA because the vascular diameter is thin. For acute-phase revascularization in particular, the approach route cannot be sufficiently evaluated before surgery, suggesting that this technique is effective. In this case, the wire was guided to the contralateral VA beyond the vertebrobasilar junction, crossing over the buddy wire. Compared with the buddy wire technique in which two microguidewires are guided to the PCA, intracranial hemorrhage due to wire perforation can be avoided because the wire tip is positioned outside the cranium. In addition, the second wire does not need to be passed through the occluded lesion, which may be advantageous. A disadvantage of the cross-over technique is that guidance may be not possible when the bifurcation angle of the junction is sharp. In the present case, the wire passed through the occluded lesion was pulled back via the cross-over technique, but it was insufficient and another wire was used as a buddy wire. However, the pulled wire cannot always be re-passed through the occluded lesion. This may be solved by passing the first wire through the occluded lesion and crossing the second wire over the junction alone.

Conclusion

We report the efficacy of the cross-over buddy wire technique in which two wires were guided to the contralateral VA for revascularization of arteriosclerotic BA occlusion by coronary arterial stenting. This may be an option when a stent cannot be navigated due to vascular tortuosity and the conditions enable wire guidance to the contralateral VA.

Disclosure Statement

The authors declare no conflict of interest.

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